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The oral and dermal toxicity of selected chemicals to brown tree snakes (*Boiga irregularis*)

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Abstract

We evaluated the oral and dermal toxicity of 18 chemicals to brown tree snakes (*Boiga irregularis*). Chemicals that produced mortality when dosed orally were rotenone, propoxur, natural pyrethrins, allethrin, resmethrin, diphacinone, warfarin, and aspirin. The lowest oral doses that gave 100% mortality were: rotenone, 2.5 mg kg^{-1}; pyrethrins, 40 mg kg^{-1}; propoxur, 40 mg kg^{-1}; diphacinone, 80 mg kg^{-1}; and aspirin, 1280 mg kg^{-1}. Allethrin, resmethrin, and warfarin produced 80% mortality at 40 mg kg^{-1}, the highest dose tested. Materials given orally that produced little mortality were permethrin, fenvalerate, and carbaryl; those giving no mortality were phenothrin, tetramethrin, piperonyl butoxide, propylene glycol, and cholecalciferol. Chemicals that produced mortality when applied dermally at doses of 40 mg kg^{-1} were rotenone, nicotine, propoxur, natural pyrethrins, allethrin, and resmethrin; those that gave no mortality were permethrin, fenvalerate, phenothrin, tetramethrin, piperonyl butoxide, and diphacinone. Rotenone, at 10 mg kg^{-1}, and nicotine, at 40 mg kg^{-1}, were the most toxic dermally, killing all tested snakes. Piperonyl butoxide enhanced the oral toxicity of allethrin and resmethrin and the dermal activity of resmethrin; it did not enhance the activity of natural pyrethrins either orally or dermally.

Introduction

The brown tree snake (*Boiga irregularis*) is a significant and invasive exotic pest that was inadvertently introduced on Guam from New Guinea after World War II (Fritts 1988). This snake was detected on Guam in the 1950s, became conspicuous in the 1960s, and is presently distributed throughout the island, with population densities estimated to be several thousand per square mile in some areas. The brown tree snake, which is arboreal and nocturnal, has probably been the primary factor for the extirpation or drastic reduction in population of several bird species on Guam (Savidge 1987): the Guam rail, the Guam flycatcher, the Micronesian honeyeater, the Micronesian kingfisher, the Mariana fruit-dove, white-throated ground-dove, and the bridled white-eye. Brown tree snakes are also agricultural pests, killing chickens, pigeons, caged song birds, newborn pigs, kittens and puppies (Fritts and McCoid 1991).

They are also nonagricultural pests on Guam. Snakes gain access to electrical transmission lines and transformers by climbing guy wires and power poles. Electrical faults are created when snakes simultaneously touch live and grounded conductors, causing power failures which interrupt electrical power use by public, commercial, and military activities (Fritts *et al.* 1987). Since 1978, more than 1200 snake-related power failures were reported by the US Navy and Guam Power Authority. The exact cost of these power failures from loss of work productivity, damage to electrical power equipment, domestic appliances, commercial equipment, and perishable items, and the labour to restore power is unknown, but losses are estimated to be in the millions of dollars.

The brown tree snake is a public health risk. A 2-year review of 94 snakebite cases in Guam showed a high proportion (80%) of victims were bitten while asleep in their homes (Fritts *et al.* 1994). Of children less than 1 year old who were bitten, 82% comprised infants 1–3 months old. The incidence of bites averaged 3.5 per month during the 2 years. The brown tree snake is mildly venomous and must chew on its victim to inject venom; adults being bitten can easily

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remove the snake, but small children may have difficulty. Infants and small children have experienced severe reactions to the bites, but no deaths have been recorded.

Guam is a focal point of air and ship cargo traffic in the Pacific, and there is the threat that brown tree snakes could accidentally be introduced to snake-free islands in the Pacific through shipments of cargo. Brown tree snakes have been found in other Pacific regions (Honolulu, Hawaii; Kwajalein Island; Wake Island; Pohnpei; and Saipan), but apparently Guam has the only breeding population outside of its native range (Fritts 1987).

Currently on Guam, the brown tree snake control programme relies on the use of live-traps to capture snakes in high-priority areas where cargo is stored or shipped from and the use of terrier dogs to detect and interdict snakes in cargo, on shipboard, or in aircraft due for departure from Guam to other Pacific ports or ports in mainland Asia and North America. Our purpose in screening chemicals for their toxicity to brown tree snakes was to try to develop additional tools for their use in an integrated snake-management programme on Guam.

There are several reports, mostly anecdotal, indicating that some of the commercial pyrethrin- or pyrethroid-containing products registered as insecticides by the US Environmental Protection Agency (EPA) are toxic to snakes. The natural and synthetic pyrethroid chemicals have good safety records and low toxicity to mammals; however, the synthetic pyrethroids are more toxic to aquatic organisms (Coats *et al.* 1989; Eisler 1992). Savarie and Bruggers (1992) concluded that pyrethrins and synthetic pyrethroids offered some of the best oral and dermal candidate toxicants for brown tree snakes. Kihara and Yamashita (1978) used Oshima lizards *(Eumeces oshimensis)* as experimental surrogates for the habu *(Trimeresurus flavoviridis)*, a venomous snake found in Okinawa, to determine that a pyrethrin was an effective dermal toxicant. Toriba *et al.* (1992) reported that a commercial aerosol product containing pyrethroids killed snakes in a few hours when applied dermally. Recently, Brooks *et al.* (in press) demonstrated that some commercially available aerosol insecticides containing pyrethrins or pyrethroids were lethal when applied dermally to brown tree snakes.

There is abundant evidence that the snake epidermis in many species is permeable to water gain and loss and that it is not necessary for snakes to drink free water in order to maintain their water balance (Bentley and Schmidt-Nielson 1966; Prange and Schmidt-Nielson 1969; Dunson and Robinson 1976; Bentley 1976; Dunson 1979). The flux of water through the skin is controlled by the layers of lipids in the mesos layer of the epidermis (Roberts and Lillywhite 1980, 1983). Since water easily passes through the epidermis in both directions, it is reasonable to suspect that other chemicals, whether dissolved in water, ethanol or other solvents, may pass readily through the snake epidermis.

Other chemicals may be orally or dermally toxic to brown tree snakes, including rotenone, nicotine, propoxur, carbaryl, diphacinone, warfarin and aspirin. The objective of this study was to assess the oral and dermal toxicity of these selected chemicals to brown tree snakes as part of a research project to develop methods to be used in a brown tree snake management programme on Guam.

Materials and Methods

This study was conducted on Guam using brown tree snakes that were live-trapped on the island during operational snake control by USDA/Animal Damage Control personnel. The snakes were transferred from traps into either wire holding cages or into cloth bags for transport to the holding facility located at Andersen Air Force Base (AAFB). Snakes were caged individually in plastic storage boxes that measured 13″ × 8.25″ on the bottom, 15.25″ × 11″ on the top, 9″ high, and contained 14 airholes (⅛″ in diameter) on the sides. We assigned each snake a unique accession number. In each cage we placed a sheet of newspaper and a double-walled plastic dish with a hole cut into the side; this dish served as a water container and shelter for the snake. Water and paper were changed weekly. Snakes were not fed either before or after being tested. After caging, snakes were quarantined for a minimum of 3 days and then examined by a veterinarian. Only animals with normal appearances (aggressively striking, no wounds, using the shelters) were allocated to the experiments.

The chemicals screened for toxicity were:

Natural pyrethrins	CAS # 8003-34-7
Allethrin	CAS # 584-79-2
Fenvalerate	CAS # 51630-58-1
Permethrin	CAS # 52645-53-1
Phenothrin	CAS # 26002-80-2
Resmethrin	CAS # 10453-86-8
Tetramethrin	CAS # 7696-12-0
Piperonyl butoxide	CAS # 51-03-6
Warfarin	CAS # 81-81-2
Diphacinone	CAS # 82-66-6
Cholecalciferol	CAS # 67-97-0
Aspirin	CAS # 50-78-2
Propoxur	CAS # 114-26-1
Carbaryl	CAS # 63-25-2
Rotenone	CAS # 83-79-4
Nicotine	CAS # 54-11-5
Propylene glycol	CAS # 57-55-6
Ethanol	CAS # 64-17-5

All chemicals, with the exception of aspirin, propylene glycol and ethanol, are registered as pesticides with the EPA.

Twelve chemicals were evaluated dermally and 17 orally. Chemicals were preweighed into 50-mL bottles and were dissolved or suspended by adding the appropriate amounts of ethanol (95%) or propylene glycol before use. In order to determine the effects of piperonyl butoxide, we added this material to the pyrethrins and pyrethroids at five times the concentration of the active ingredient. We did the study in two phases. The first phase used ethanol as a carrier for orally and dermally applied toxicants; the second phase used propylene glycol as the carrier for orally dosed toxicants. Ethanol was used in the first phase because most of the chemicals were soluble in it. It did cause some temporary intoxication and lethargy of the orally-dosed snakes, however. This prompted the use of propylene glycol as the oral carrier in the second phase.

Snakes were allocated randomly to treatment groups by body weight. Five snakes of either sex were used in each treatment. We dosed snakes orally and dermally using Hamilton7 precision syringes accurate to 0.01 mL. A ball-tipped feeding needle (18 gauge, 1½″ long) was used on a syringe, and oral doses were administered by inserting the needle into the entrance of the oesophagus. These same ball-tipped needles were used to apply dermal doses to the lateral and ventral surfaces from the neck to the vent of snakes while they were physically restrained. The volume of carrier and toxicant administered was 1 mL per 100 g body weight for both oral and dermal dosing.

Snakes dosed with pyrethrins, pyrethroids, propoxur, carbaryl, rotenone, nicotine, piperonyl butoxide or aspirin were returned to their cages and observed for 72 hours for signs of intoxication or mortality. Snakes dosed with anticoagulants or cholecalciferol were observed for 7 days (this interval was based on that seen in poisoned mammals). All snakes that survived the observation period were euthanased with halothane (Andrews *et al.* 1993) and disposed of by deep burial in a sanitary landfill. All procedures were approved by the NWRC Institutional Animal Care and Use committee.

Results

Dermal Toxicity

Six of the 12 chemicals (allethrin, resmethrin, pyrethrins, propoxur, nicotine and rotenone) produced mortality in brown tree snakes at a dermal screening dose of 40 mg kg^{-1} (Table 1). The synthetic pyrethroids (permethrin, fenvalerate, phenothrin and tetramethrin), and diphacinone showed no dermal toxicity. Snakes were treated with 20 mg kg^{-1} doses of allethrin, resmethrin, pyrethrin, and propoxur; allethrin and pyrethrin produced some mortality, resmethrin and propoxur did not. Lower doses of the other synthetic pyrethroids were not tested. Piperonyl butoxide dosed at 80 mg kg^{-1} gave no signs of intoxication or mortality. Nicotine and rotenone were the most toxic of the dermally-tested chemicals; nicotine gave complete kills at 40 mg kg^{-1} and rotenone killed all snakes at 10 mg kg^{-1}. All control snakes dosed with ethanol survived.

Table 1. Mortality of brown tree snakes when dosed dermally with several toxicants, using ethanol as the carrier
(No. dead/No. treated)

Toxicant	Dose (mg kg^{-1})						
	0	2.5	5	10	20	40	80
Permethrin	–	–	–	–	–	0/5	–
Fenvalerate	–	–	–	–	–	0/5	–
Phenothrin	–	–	–	–	–	0/5	–
Tetramethrin	–	–	–	–	–	0/5	–
Piperonyl butoxide	–	–	–	–	–	–	0/5
Diphacinone	–	–	–	–	–	0/5	–
Resmethrin	–	–	–	–	0/5	1/5	–
Pyrethrins	–	–	–	–	1/5	2/5	–
Allethrin	–	–	–	–	2/5	3/5	–
Propoxur	–	–	–	–	0/5	3/5	2/5
Nicotine (Free–base)	–	–	–	0/5	3/5	5/5	–
Rotenone	–	0/5	2/5	5/5	5/5	5/5	5/5
Control (Ethanol)	0/15	–	–	–	–	–	–

Oral Toxicity

Permethrin and fenvalerate killed a few snakes when given orally with ethanol as the carrier (Table 2). The pyrethrins, allethrin and resmethrin, proved to be orally lethal to brown tree snakes; pyrethrins were the most toxic, killing some snakes in doses as low as 10 mg kg^{-1}. Diphacinone also killed snakes within 24 h in doses of 10–40 mg kg^{-1}. Warfarin killed snakes at 40 mg kg^{-1} but they died slowly, one 7 days after dosing. None of the anticoagulant materials produced signs of haemorrhage in the snakes.

On the second screening of candidate toxicants, using propylene glycol as the carrier, rotenone was the most toxic of the materials tested, followed by natural pyrethrins and propoxur

Table 2. Mortality of brown tree snakes when dosed orally with several toxicants, using ethanol as the carrier
(No. dead/No. treated)

Toxicant	Dose (mg kg^{-1})		
	10	20	40
Allethrin	–	2/5	4/5
Resmethrin	–	2/5	4/5
Pyrethrins	1/5	4/5	5/5
Permethrin	–	0/5	2/5
Fenvalerate	–	–	1/5
Phenothrin	–	–	0/5
Tetramethrin	–	–	0/5
Piperonyl butoxide	–	–	0/5
Diphacinone	1/5	1/5	5/5
Warfarin	–	–	4/5
Cholecalciferol	–	–	0/5
Control (Ethanol)	–	–	0/5

(Table 3). Rotenone killed all snakes at 2.5 mg kg^{-1}. Diphacinone was lethal at doses of 20 mg kg^{-1} and greater; with complete kills occurring at 80 mg kg^{-1}. Warfarin failed to kill any snakes at 20 and 40 mg kg^{-1} when given with propylene glycol and was not tested further. Carbaryl killed 1 snake (of 5) at both doses tested (80 and 160 mg kg^{-1}) and was not further tested. Aspirin was toxic and lethal at doses of 640 and 1280 mg kg^{-1}. Propylene glycol was not toxic.

Poisoning Symptoms

Rotenone killed snakes in 37–55 min at the higher doses. Symptoms of poisoning observed were a gaping mouth and extreme lethargy beginning at 28 min and laboured respirations every 65–75 s, seen at 34 min in one snake. The snakes gave the appearance of gasping for breath and being in respiratory distress.

Propoxur produced poisoning symptoms as soon as 38 min after dosing; snakes lost their righting reflex, became extremely lethargic, and, at 40 mg kg^{-1} doses, were dead by 39–46 min. We observed some snakes with frothy liquid in their oesophagus and with gaping mouths; some had copious secretions from the mouth at death. Carbaryl exhibited similar symptoms, affecting snakes in 17–40 min after dosing; they exhibited extreme lethargy or a moribund state, only to recover 24–48 h later.

Pyrethrins and pyrethroids affected snakes by inducing general muscular tremors and disorientation, leading to a moribund condition, often with the snake lying ventral side up. Death sometimes occurred within a few hours and usually within 24–48 h. The eyes were often completely dilated. Tetramethrin produced overt poisoning symptoms, such as generalised muscular tremors, disorientation, loss of the 'righting reflex' (with snakes remaining on their backs), and a moribund condition within 1–2 h after dosing; yet the snakes recovered without mortality by the end of 48 h.

Diphacinone at 40–80 mg kg^{-1} oral doses killed snakes overnight with no apparent symptoms; snakes died in a relaxed position. At necropsy, there was no evidence of haemorrhage in the body tissues. Warfarin, dosed with ethanol, also produced death in snakes as long as 7 days after dosing. No apparent poisoning symptoms or haemorrhages were noted. When warfarin was dosed with propylene glycol, no mortality was observed.

Nicotine killed snakes overnight. No overt symptoms of intoxication were observed; snakes simply appeared to die in a relaxed state.

We saw no overt symptoms of intoxication with aspirin. Dead snakes appeared relaxed with no signs of convulsions.

Table 3. Mortality of brown tree snakes when dosed orally with several chemicals, using propylene glycol as the carrier
(No. dead/No. treated)

Toxicant	Dose (mg kg^{-1})											
	0.61	1.25	2.5	5	10	20	40	80	160	320	640	1280
Rotenone	0/5	1/5	5/5	5/5	5/5	5/5	5/5	–	–	–	–	–
Pyrethrin	–	–	–	0/5	4/5	3/5	5/5	–	–	–	–	–
Propoxur	–	–	–	0/5	2/5	3/5	5/5	–	–	–	–	–
Diphacinone	–	–	–	–	0/5	1/5	3/5	5/5	–	–	–	–
Warfarin	–	–	–	–	–	0/5	0/5	–	–	–	–	–
Carbaryl	–	–	–	–	–	–	1/5	1/5	–	–	–	–
Aspirin	–	–	–	–	–	–	–	–	0/5	0/5	3/5	5/5
Controls	–	–	–	–	–	–	0/5	–	–	–	0/3	–

Piperonyl butoxide potentiation

Piperonyl butoxide, given orally along with pyrethroids and pyrethrins appeared to enhance the mortality in three of the four materials tested (Table 4). It did not enhance natural pyrethrins; in fact, a lower mortality was obtained than with the compound alone. In dermal toxicity tests, piperonyl butoxide was tested with allethrin, resmethrin, and pyrethrins; mortality appeared to be increased only in resmethrin, while it appeared to decrease mortality in the other materials.

Table 4. Effects of piperonyl butoxide[A] (P.b.) as a synergist with pyrethrins and pyrethroids inbrown tree snakes, using ethanol as the carrier
(No. dead/No. treated)

	Mortality	
Toxicant	Oral dose 20 mg kg^{-1}	Dermal dose 40 mg kg^{-1}
Allethrin	2/5	3/5
Allethrin + piperonyl butoxide	4/5	1/5
Resmethrin	2/5	1/5
Resmethrin + piperonyl butoxide	4/5	5/5
Pyrethrins	4/5	2/5
Pyrethrins + piperonyl butoxide	2/5	1/5
Permethrin	0/5	–
Permethrin + piperonyl butoxide	1/5	–

[A]Piperonyl butoxide was present at 5 times the concentration of the pyrethrins/pyrethroids, i.e. 100 mg kg^{-1} orally and 200 mg kg^{-1} dermally.

Discussion and Conclusions

Dermal Toxicity

Since water easily passes through the snake epidermis in both directions, it is reasonable to suspect that other chemicals, whether dissolved in water, ethanol or other solvents, may pass readily through the epidermis. It was apparent from the previous aerosol insecticide study that toxicants readily penetrated the skin of brown tree snakes (Brooks *et al.*, in press), where some signs of intoxication were seen within 1 h when snakes were sprayed with aerosol insecticides. The dermal application of six of the chemicals dissolved in ethanol caused death in snakes in doses of 40 mg kg^{-1} or less in the case of rotenone. Of all chemicals tested, rotenone and nicotine showed promise for further research as dermal toxicants.

Oral Toxicity

Rotenone was the most toxic of the chemicals tested orally, killing brown tree snakes in doses as low as 2.5 mg kg^{-1}. Rotenone kills by oxygen deprivation (Fontenot *et al.* 1994), through the blocking of reoxidation of reduced nicotinamide adenine dinucleotide by NADH-dehydrogenase in the mitochondria (Horgan *et al.* 1968; Lindahl and Oberg 1961; Oberg 1961). The treated snakes suffocated and died in less than an hour in most cases. Rotenone is an unstable compound that is nonpersistent and essentially does not bioaccumulate (Fontenot *et al.* 1994). It is hazardous to fish, amphibians, and some reptiles, and could be a concern of possible poisoning of nontarget species if used in aquatic and some terrestrial habitats.

Pyrethrins, allethrin, and resmethrin are photo-labile and degrade in the presence of sunlight into relatively less active and possibly less toxic substances (Otieno and Pattenden 1980; Leahey

1985). This presents both advantages and disadvantages in the field: baits containing these materials exposed to direct sunlight would have a very limited activity period before degradation. This could be desirable and could be prevented by exposing baits in darkened containers. Pyrethrins/pyrethroids could be expected to be toxic to non-target reptiles, such as geckos, skinks, and lizards.

We found that propoxur was almost as toxic to brown tree snakes as natural pyrethrins and killed snakes much faster. An aerosol wasp and hornet killer containing propoxur was quickly lethal to brown tree snakes (Brooks *et al.*, in press). We saw symptoms of propoxur poisoning (loss of righting reflex) as soon as 38 min after dosing. Propoxur is degraded fairly rapidly in water (Eichelberger and Lichtenberg 1971) and probably does not bioaccumulate significantly in fish, aquatic organisms, and cattle tissues or milk (Stanley and Thornton 1972). It may be a concern for possible poisoning of non-target species of other reptiles (geckos, skinks, and lizards) on Guam.

Diphacinone killed all 5 snakes overnight when given orally in doses of 40–80 mg kg^{-1}; no snakes were killed when it was given dermally at 40 mg kg^{-1}. It may have value as a toxicant for oral delivery systems for brown tree snakes. Warfarin given orally at 40 mg kg^{-1} in ethanol killed 4 of 5 snakes slowly (mean 4.5 days). However, when gavaged at 40 mg kg^{-1} using propylene glycol as the carrier, no snakes died. No signs of haemorrhage were seen (at necropsy) in snakes that died from either anticoagulant material. Observations of convulsions and death without haemorrhagic signs were recorded by Hagan and Radomski (1953) in several species of laboratory animals (rats, monkeys, dogs, rabbits, guinea pigs, and chickens) that died within several hours of being gavaged with high doses of warfarin. Recently, Jolly *et al.* (1994) noted that pindone (closely related to diphacinone) caused death in possums without haemorrhage, apparently by liver damage. These observations suggest that in case of acute toxic doses, some mechanisms other than inhibition of vitamin K–dependent clotting factors may be occurring.

Aspirin, which was toxic and lethal only at high doses, is worth considering as an oral snake toxicant. At levels of =300 mg in individual baits, aspirin could be used in the field. The LD_{50} of aspirin to mammalian species is fairly high – about 1100–1500 mg kg^{-1} for mice and rats (Hart 1947) – making it relatively innocuous for dogs, pigs and humans (human lethal dose is 20–30 g: Wenger and Einstein 1970). Cats, however, are quite susceptible to its effects. Cats metabolise aspirin very slowly in comparison with dogs (Yeary and Swanson 1973), and aspirin in cats can cause anemia and bone marrow hypoplasia (Penny *et al.* 1967). Aspirin is not registered with the EPA as a vertebrate toxicant, however.

Exposure of any toxicants in the field, either for oral or dermal delivery to brown tree snakes, will probably be done in some kind of covered container or toxicant-delivery device. These can be designed to exclude adults of the large introduced monitor lizard, *Varanus* species, on Guam, but will not keep out the smaller reptiles and juvenile monitor lizards. It is hoped that baits attractive to brown tree snakes will not be attractive to the insectivorous geckos and lizards. Toxicant containers will probably be placed off the ground into the vegetation, as are live-traps, to help in keeping them from the reach of dogs and pigs.

This study demonstrates that certain classes of chemicals (mainly botanicals) are orally and dermally toxic to brown tree snakes. Snake skin permits the fairly rapid passage of toxicants, as seen in the insecticide aerosol study (Brooks *et al.*, in press), where some signs of poisoning were seen within 1 h of spraying. Delivery of toxicants to brown tree snakes by both the oral and dermal routes remains to be explored in the near future.

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